equally apply to management of perfusion pressure during cardiac operations as well.

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12/8/73103

Reply to the Editor:

The members of the Cornell Coronary Artery Bypass Outcomes Trial Group (CCABOT) thank Drs. Keats and Slogoff for their insightful comments and appreciate the opportunity to expand on the points that they have raised in their letter regarding our article.[1] The trial was designed as a prospective randomized study at the outset. The criteria for major and minor outcomes were set a priori. The principal outcomes were as follows: all cause mortality, cardiopulmonary morbidity (myocardial infarction, pulmonary edema, adult respiratory distress syndrome, cardiogenic shock/low flow state, and cardiopulmonary arrest), neurologic morbidity (new major focal deficit), cognitive complications (defined by intrapatient deterioration on neurocognitive tests), and deterioration in functional status (decline on the SF-36 health survey). Minor outcomes were also determined a priori and were not included as principal outcomes. The minor cardiac outcomes included myocardial ischemia and congestive heart failure and the minor neurologic outcomes included focal deficit lasting less than 24 hours. All major and minor outcomes were counted up to the 6-month interval after the operation and were stipulated before the start of the trial.

When the major outcomes were analyzed at the completion of the trial, it was found that patients randomized to the low mean arterial pressure (MAP) group had a higher incidence of all cause death, major cardiac complications, and major neurologic complications. The combined incidence of total mortality and major cardiac and neurologic morbidity was 12.9% in the low MAP group versus 4.8% in the high MAP group ($p = 0.026$). The incidence of deterioration in neurocognitive function and deterioration in functional status did not differ between the two groups.

The mean arterial pressures between the low and high MAP groups differed by an average of 18 mm Hg. As shown in Table VI (the pragmatic analysis), which demonstrates the actual pressure achieved regardless of randomization group, the trend toward lower complications with higher pressures can clearly be seen.

Although it is tempting to ascribe differences in outcome to variables other than the therapeutic maneuver, which was assigned by randomization, it is extremely unlikely that the pattern of specific variables differed appreciably between the two MAP groups. The very raison d'être of a randomized trial is the statistical balance of potential confounding factors, both those previously identified and those unknown at the inception of the trial. Table I of the article demonstrates this balance for a large number of such variables. Furthermore, although not incorporated into the paper, many additional factors were analyzed to ascertain whether there were any differences between the two MAP groups, either before or after bypass. All of these items, including MAPs, cardiac outputs, anesthetic agents, vasoactive medications, other medications, blood gases, activated clotting times, hematocrit values, and other hemodynamic parameters were not found to differ between the two treatment groups.

Pilot subsets of patients enrolled in the present trial also underwent preoperative transesophageal echocardiogram and intraoperative transcranial Doppler ultrasonography; because these subsets were selected after randomization and were examined in a nonrandom fashion, they cannot yield unbiased information on the importance of atheromatous disease or embolic load for the entire 248-patient cohort randomized to two treatment arms. Because atheromatous disease of the aorta is now thought to be a risk factor for perioperative complications of coronary bypass,[2] the reproducible quantification of aortic atheroma by transesophageal echocardiography is under development.[3] Future clinical trials of coronary bypass will incorporate these procedures in all patients.

Animal and human data support the conclusion that autoregulation of cerebral blood flow occurs across the range of MAPs of 50 to 150 mm Hg. As observed, 54 mm Hg falls within this range. However, the data also suggest that the autoregulatory range of cerebral blood flow is shifted to the right in patients with longstanding hypertension. In addition, these conclusions are based on experiments in which global cerebral blood flow was measured during the conditions of normothermic pulsatile perfusion. Extension of these conclusions to the conditions of nonpulsatile, hypothermic bypass with attention to regional blood flow may not be valid. Patients with coronary artery disease are known to have an increased incidence of other vascular disease, including that of the cerebral circulation. Higher perfusion pressures may permit the maintenance of adequate blood flow to areas distal to stenotic vessels or facilitate recruitment of collateral blood flow, thus minimizing the affected area of ischemia.

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12/8/73104

Mitral valve repair

To the Editor:

My colleagues and I read with interest the article by Hvass and associates (J Thorac Cardiovasc Surg 1995;110: 859-62), in which they described a technique for mitral valve repair that closely resembled a procedure we described in this Journal in 1994 (1994;107:635-8). We both described a technique of grafting the posterior leaflet of the tricuspid valve, with the chordae tendineae and papillary muscle, to the mitral valve leaflets for the correction of mitral valve insufficiency.

To our surprise, the authors made no reference to our work in their article. Inasmuch as our letter was published in the same Journal, we thought that it could not have escaped the attention of either the authors or the referees.

We would also like to draw attention to the fact that we suggested further use of this technique for partial replacement of the leaflets in the case of localized calcification of the mitral valve.

We are glad that Dr. Hvass and his group have confirmed the efficiency of this technique, which we have now used successfully in 20 cases.

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12/8/72896

A simple switching technique from cardiopulmonary bypass to a long-term extracorporeal life support system

To the Editor:

We read with great interest the article by Muehrcke and associates[1] in the September 1995 issue of the Journal.[1] They summarized their clinical experiences with an extracorporeal life support (ECLS) system and reported that limb ischemia was the most common complication, found in 70% of patients who underwent percutaneous ECLS. Besides limb ischemia, bleeding from the surgical wound is also a life-threatening complication of ECLS.[1, 2] Since 1965 we have used ECLS in more than 60 patients with severe cardiopulmonary failure.[3, 4] To overcome the aforementioned problems, we recently began routinely using the following technique (Fig. 1).

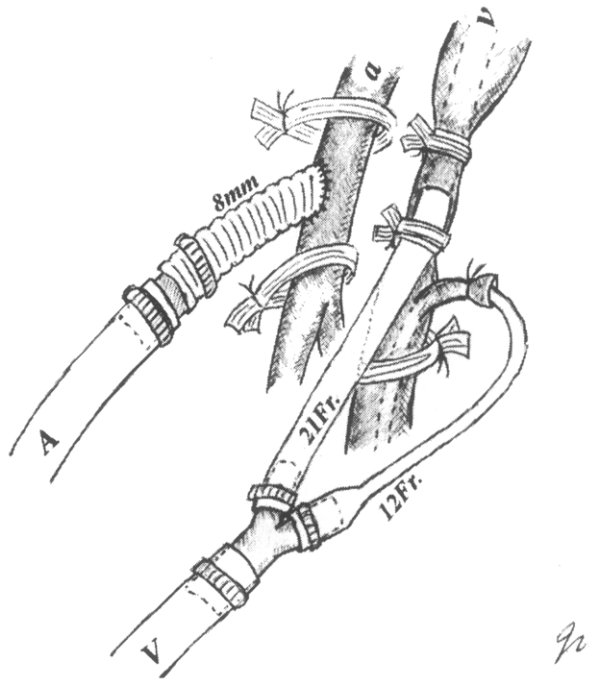

Fig. 1. A cannulation technique to switch from CPB to ECLS for postcardiotomy cardiac failure. A Dacron vascular graft is sutured to the right femoral artery. A 12F wire-reinforced cannula is inserted through the saphenous vein to the distal femoral vein. A 21F heparin-bonded cannula is inserted through the femoral vein to the right atrium. The lines are connected to a heparin-bonded ECLS system. The CPB flow is decreased and the ECLS flow is increased to allow the switch. *A,* artery; *V,* vein.

When a patient is difficult to wean from cardiopulmonary bypass (CPB) because of cardiac failure, an intraaortic balloon pump is initially inserted in the left femoral artery after an 8 mm sealed Dacron prosthesis has been sutured to the left femoral artery in an end-to-side fashion. The intraaortic balloon pump is started in a 1:1 mode. The ECLS is set up during CPB. The entire system is heparin-bonded and composed of a capillary membrane oxygenator (Maxima: CB1380, Medtronic Inc., Cardiopulmonary Div., Anaheim, Calif.), a Bio-Medicus model 540T centrifugal pump (Medtronic Bio-Medicus, Eden Prairie, Minn.), and tubes and connectors. Our switching procedure is illustrated in Fig. 1. The right femoral artery and vein are taped. A second 8 mm sealed Dacron prosthesis is sutured to the femoral artery in an end-to-side fashion and then connected to the arterial line of the ECLS system. A 12F wire-reinforced cannula is inserted through the saphenous vein to the distal femoral vein and fixed with 3-0 silk. The CPB flow is decreased 50% and the inferior vena caval cannula used for CPB is removed. A 21F heparin-bonded wire-reinforced cannula with multiple side holes (Medtronic) is inserted through the femoral vein to the right atrium and fixed with two Teflon tapes with double ligation. The two venous cannulas are con-